Schizotypy: Looking Back and Moving Forward

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Abstract:

This article introduces and reviews the history of the construct of schizotypy for the special section appearing in the journal. Schizotypy offers a useful construct for understanding the etiology, development, and expression of schizophrenia-spectrum psychopathology and a unifying construct for linking a broad continuum of clinical and subclinical manifestations. The article reviews the descriptive psychopathology roots of schizotypy, Meehl and Claridge's classical formulations of the construct (including the debate about dimensional vs taxonic structure), and the need for a comprehensive, multidimensional model of schizotypy. The article briefly reviews the wide empirical literature supporting schizotypy and also examines several criticisms and misconceptions about the construct and research methods used to assess it. Finally, the article offers several suggested goals for future schizotypy research.

Keywords: schizophrenia | schizotypal | psychosis proneness

Article:

Overview

Schizotypy offers a useful and unifying construct for understanding schizophrenia-spectrum psychopathology. Useful in that it has explanatory power for understanding the development, expression, trajectory, risk and resilience, and treatment of schizophrenia-spectrum conditions, as well as for understanding variation in normal behavior, and unifying because it encompasses a broad spectrum of conditions—schizophrenia, related psychotic disorders, spectrum personality disorders, the prodrome, and subclinical expressions—under a single conceptual framework. Furthermore, schizotypy allows us to examine the etiological and developmental pathways underlying schizophrenia-spectrum psychopathology while minimizing many of the confounding effects that complicate the study of patients. However, the construct of schizotypy has suffered from the lack of a clear identity, as well as from conflicting identities in the literature, and the term is often used interchangeably with a variety of other descriptors. Nevertheless, schizotypy is an increasingly studied construct with a recent PsychInfo search (completed July 22, 2014) of the term indicating more than 1100 publications (including more than 400 since 2010). This brief review traces the history of schizotypy, differentiates it from related constructs, attempts to offer

an integrative definition, addresses criticisms regarding the construct, and offers recommendations for future studies of schizotypy.

Brief History of Schizotypy

The term schizotypy was introduced more than 60 years ago to describe a broad phenotype of schizophrenic–like psychopathology and impairment. However, the origins of schizotypy date back at least another half century, and drew from both clinical and personality traditions. In the clinical domain, Kraepelin[1] and Bleuler[2] described schizophrenic–like traits in patients prior to their illness and in the relatives of patients. Kraepelin described these mild and subclinical symptoms as precursors to dementia praecox. However, he also suggested that psychotic–like experiences in relatives could signify an arrested form of the illness. Bleuler vividly described that "entirely crazy acts in the midst of normal behavior" can precede the development of schizophrenia[2] (p. 252). The descriptive psychopathology literature includes numerous reports that mild forms of schizophrenia often appear in the nonpsychotic relatives of patients[3–6] and precede the onset of clinical psychosis, although they often represent stable expressions that do not advance into full-blown disorders.[7,8] Furthermore, the early and mid-20th century offered a number of schizophrenic–like conditions such as borderline, simple, ambulatory, and pseudoneurotic schizophrenia that captured characteristics of schizotypy.[9]

Rado[10] initially introduced the term schizotype to represent the schizophrenic phenotype, based upon his observations that there was a continuum of schizophrenic–like behavioral impairment. He indicated that the liability for schizophrenia was genetically driven and that this vulnerability resulted in impairment ranging from mild to fully schizophrenic. Meehl[11,12] posited that a single dominant "schizogene" (in conjunction with other genetic potentiators) produced a neurointegrative defect referred to as schizotaxia that was necessary, although not sufficient, for the development of schizotypy (and by extension, schizophrenia). He viewed schizotypy as the personality organization that resulted from schizotaxia and conveyed vulnerability for the development of schizophrenia. Meehl's[13] checklist provided a rich description of schizotypic signs and symptoms such as cognitive slippage, anhedonia, and magical ideation. He substantially updated his original model by introducing hypokrisia (a neuronal level aberration that characterizes schizotaxia), diminishing the role of anhedonia, and expanding the impact of cognitive slippage and the contribution of polygenetic potentiators.12 Note that Lenzenweger[14] provided a comprehensive review of Meehl's model of schizotypy.

Meehl described that schizotypy is taxonic in nature, suggesting that approximately 10% of the population is schizotypic and that about 10% of schizotypes decompensate into schizophrenia (arriving at the 1% lifetime prevalence rate of schizophrenia). However, his taxonic formulation of schizotypy was based upon his single-gene model of schizotaxia, schizotypy, and schizophrenia that ultimately has not received support in the literature, eg,[15,16], which currently conceptualizes schizophrenia as a complex, heterogeneous, and polygenic disorder (or family of disorders).[17] Note that Meehl viewed that polygenetic potentiators could increase or decrease the likelihood of a schizotypic individual developing schizophrenia, but at the core of his model was a single gene that was necessary, albeit not sufficient, for schizophrenia to develop. More than a dozen studies have employed taxometric analyses to study the structure of schizotypy primarily employing questionnaire measures. The majority of these studies, eg,[18,19] provided at least partial

support for a schizotypy taxon (or taxa) with base rates in nonclinical samples generally approximating Meehl's 10% estimate; although there have been notable exceptions.[20,21] Widiger[22] raised concerns about taxonic models of psychopathology given that mental disorders likely result from multifactorial genetic and nongenetic origins that are inconsistent with such models. Thus, support for taxonic models of schizotypy requires a compelling etiological conceptualization that provides a basis for discontinuity, not simply results of taxometric analyses. Edmundson and Kwapil[23] discussed that the heterogeneity of schizotypy and schizophrenia also raises questions about the extent to which separate etiological processes are occurring and how many taxa may be represented in our current conceptualization of schizotypy. The issue of whether schizotypy is taxonic remains to be resolved, but we suggest that the search for a schizotypy taxon (or taxa) should be predicated on an etiological model that supports a discontinuity in nature.

Lenzenweger[14,24] has been a leading proponent of Meehl's conceptualization that schizotypy is the latent personality organization that conveys liability for schizophrenia. He has supported the role of genetically driven schizotaxia, or neurodevelopmental disruptions, giving rise to schizotypy and argued for the role of secondary biopsychosocial "hits" that exacerbates risk for clinical manifestations.[25] Lenzenweger[24] described that schizotypy can be identified by familial/biological, clinical, and psychometric-laboratory index approaches.

The study of schizotypy also derived from the field of individual differences. Claridge and colleagues, eg, [26,27] offered an alternative model of schizotypy that was built upon dimensional models of personality and psychopathology and conjectured that schizotypy is fully dimensional in nature and includes adaptive manifestations (see figure 1). Claridge contrasted his fully dimensional model with Meehl's "quasi-dimensional" approach that viewed expressions of schizotypy as formes frustes of disease. Claridge's model proposes that schizotypy results from a combination of genetic, environmental, and personality variations that are normally distributed in the general population. Like Meehl's formulation, the fully dimensional model recognizes dimensionality of schizotypy in the clinical and subclinical ranges, but also argues for continuity of schizotypic traits that are part of normal individual differences expressed in the general population. Thus, Claridge argues that it includes the pathological, quasi-dimensional components, but also encompasses healthy manifestations (eg, creativity). Claridge and Beech[27] suggested that schizotypy can be thought of as analogous to trait anxiety that has expressions in the general population, but in its extreme forms results in clinical disorders.

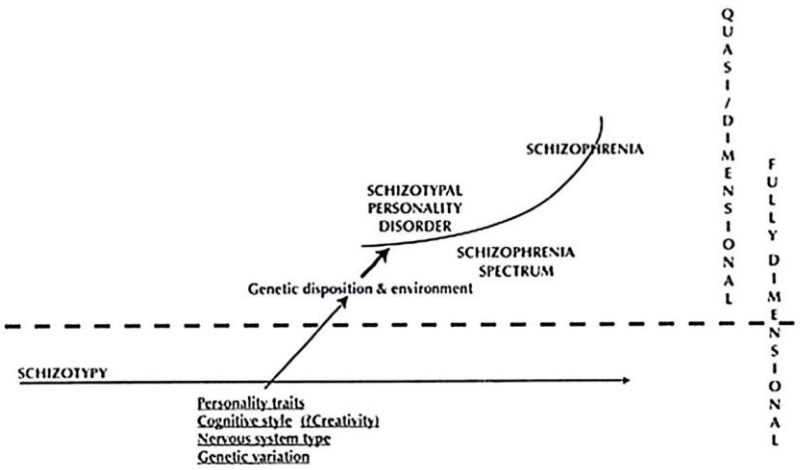

Figure 1. Diagram of the fully dimensional model of schizotypy from Claridge and Beech[27].

Schizotypy as a Multidimensional Construct

Schizotypy and, by extension, schizophrenia are heterogeneous. This heterogeneity is apparent at the phenotypic level, with symptoms and impairment ranging from mild disruptions (eg, odd beliefs, mild withdrawal) to marked excesses (eg, hallucinations) and deficits (eg, formal thought disorder). Furthermore, this heterogeneity is evident at the etiological, developmental, and treatment-response levels, and the heterogeneity of schizotypy and schizophrenia appears to be characterized by a common multidimensional structure. Factor analytic studies suggest that positive, negative, and disorganized dimensions underlie schizophrenia[28,29] and these dimensions have been replicated in nonclinically ascertained schizotypy.[30–34] The positive or psychotic–like dimension is characterized by disruptions in the content of thought (ranging from odd beliefs and magical ideation to full-blown delusions), perceptual oddities in all senses (ranging from illusions to hallucinations), and suspiciousness and paranoia. The negative or deficit dimension is characterized by diminution in experiences including alogia, anergia, avolition, anhedonia, flattened affect, and disinterest in others and the world. The disorganization dimension includes disruptions in the ability to organize and express thoughts and behavior, ranging from mild disturbances in thinking and behavior to formal thought disorder and grossly disorganized actions. Note that although there is considerable support for a three-factor model of schizotypy and schizophrenia, there is not universal agreement on the number and nature of the dimensions, and other dimensions have been proposed including a separate paranoia factor, eg,[35] and impulsive-nonconformity.[36]

The reliable identification and measurement of these dimensions is essential for parsing the heterogeneity of schizotypy and schizophrenia. In fact, treating schizotypy and schizophrenia as homogenous constructs impedes our ability to understand the origins, development, and expression of these complex conditions.[9] Consider the question of whether schizotypy is associated with substance abuse. Studies using dimensional[37] and cluster[38] approaches reported that positive schizotypy, but not negative schizotypy, is associated with elevated drug and alcohol use and impairment. Thus, failing to differentiate these dimensions and simply examining “homogenous” schizotypy suggests that conflicting results could occur depending upon the composition of the sample in terms of positive and negative characteristics. We offer

substance abuse as one example (and recognize that there are more sophisticated questions to examine regarding schizotypy and substance abuse), but this pattern of differential associations with the schizotypy dimensions arises when considering numerous psychological characteristics such as affective expression, creativity, social functioning, neuroticism, and cognitive impairment. Furthermore, it is important to consider that these dimensions may involve distinct etiological and developmental pathways, given findings of distinct disruptions in brain systems and neurotransmitter functioning in positive, negative, and disorganized symptoms in schizophrenia, eg,[39–41].

An Integrative Definition of Schizotypy for Moving Forward

Currently, there are numerous terms that are used to describe similar phenomena: schizotypy, schizotypal personality, psychosis proneness, psychotic–like experiences, anomalous experiences, as well as broad labels such as subclinical psychotic symptoms. In recent years, this terminology has expanded with descriptors of clinically indexed vulnerability for schizophrenia, such as the prodrome, basic symptoms, at risk mental states for psychosis, and attenuated psychotic symptoms syndrome recently introduced in Diagnostic and Statistical Manual of Mental Disorders (DSM)-5[42] as a condition for future research. Many of these newer syndromes are based upon attenuated forms of positive symptoms and are designed to index risk of transition into psychosis.

These terms bring added refinement to our understanding of the etiology and development of schizophrenia-spectrum psychopathology. However, we also suggest that it can reflect a lack of clarity as they are often used interchangeably with schizotypy. The utility of schizotypy requires clear operationalization (that should also drive our measurement of the construct). The inconsistent definition of schizotypy and assumed interchangeability of terms hinders our ability to integrate findings across studies. We suggest that most of the constructs represented by these other terms (eg, the prodrome) can be considered relatively narrow expressions of the schizotypy continuum and often simply represent differences in severity of symptoms. For example, non–disordered schizotypes, prodromal patients, schizotypal personality disordered patients, and psychotic patients may all experience positive symptoms such as odd beliefs, but in an increasing level of severity from magical ideation to full-blown delusions.

We posit that schizotypy is a multidimensional unifying construct that represents the underlying vulnerability for schizophrenia-spectrum psychopathology that is expressed across a broad range of personality, subclinical, and clinical psychosis phenomenology.[9] This model implies that the same etiological, developmental, and phenomenological processes underlie subclinical and clinical manifestations. Thus, schizotypy provides an ideal model for examining these processes and their development. As described above, we believe that a multidimensional conception of schizotypy is an essential aspect of this model and we suggest that a significant task for schizotypy researchers is to reach a consensus regarding this multidimensional structure.

In sum, schizotypy offers a dynamic, multidimensional model that is not constrained by diagnostic boundaries. It is not meant to replace diagnostic categories such as schizotypal personality disorder, attenuated psychotic symptoms syndrome, or schizophrenia. However, it is suggested that these categories are subsumed within the schizotypy continuum. Put differently,

schizophrenia is not considered as a separate entity from schizotypy, but rather as the most extreme expression of schizotypy. Even if there is not a universally agreed upon model of the multidimensional structure of schizotypy, it provides a useful framework for advancing the understanding of personality-psychopathology links in general and within the field of psychosis in particular, as well as for testing hypotheses concerning the etiology, risk, resilience, expression, and treatment of schizophrenia-spectrum conditions. We are not advocating that this conceptualization of schizotypy perfectly "carves nature at its joints;" however, we believe that clear operationalization is essential to guide measurement and provide the basis for construct validation.

It is worth noting that traditional categorical diagnostic systems are increasingly embracing dimensional models of psychopathology. For example, DSM-5 eliminated the differentiation of mental disorders (Axis I) from personality disorders (Axis II) given evidence that there is a common underlying structure to all psychopathology. DSM-5 offers an explicit statement about the limitations of assessing personality pathology using narrowly defined independent categories in light of the problems of high comorbidity, diagnostic status change, shared dimensional structure across categories and commonality of genetic and environmental risk factors for what were formerly assumed to be distinct disorders. Although DSM-5 formally retained the categorical model of personality disorders used in previous editions, it offers a dimensional, psychologically based model of disordered personality that employs pathological traits from five personality domains. Thus, schizotypal personality disorder is defined as a prototype characterized by impairments in identity, self-direction, empathy, and/or intimacy, along with specific maladaptive traits in the domains of psychoticism and detachment. We believe that this is a great enhancement in the conceptualization of schizotypal personality disorder, although it does not fully consider the multidimensional nature of the disorder, which is comprised of positive, negative, and disorganized features.

Support for a Multidimensional Model of Schizotypy

Despite disagreements about the construct, the lack of a clear multidimensional operationalization, and numerous and sometimes inconsistent measurement tools, cross-sectional and longitudinal studies of schizotypy have demonstrated the utility of the construct in terms of capturing variance in both clinical and nonclinical samples. The other articles in this special section review the construct validation of schizotypy in greater detail, but we offer several examples of selected findings that demonstrate the explanatory power of schizotypy. Schizotypy is presumed to convey the underlying vulnerability for schizophrenia. Therefore, non–disordered high schizotypy individuals are presumed to demonstrate mild and transient signs of the symptoms and impairments seen in schizophrenia and to be at elevated risk for transitioning into schizophrenia-spectrum disorders. Furthermore, studies have increasingly made predictions and examined findings separately for different schizotypy dimensions. Numerous studies reported that non–disordered schizotypic individuals exhibit psychotic–like,[43] prodromal,[44] schizophrenia-spectrum,[45] and basic symptoms.[46] Furthermore, longitudinal studies[47–49] demonstrated that positive schizotypy is associated with the development of psychotic disorders and negative schizotypy is associated with the development of schizophrenia-spectrum disorders. Schizotypy is also associated with schizophrenic–like patterns of cognitive impairment,[50–53] neurological soft signs,[54] and impairments on assessments of thought disorder,[55] social cognition,[56] and other

laboratory measures.[57] Schizotypy is associated with schizophrenic–like impairment on neuroscience assessments including neuroimaging,[58] EEG,[59] and eye tracking.[60] Schizotypy is also associated with distinct patterns of normal personality traits,[37] impaired attachment,[61] and creativity.[62] In addition, recent studies have employed experience sampling methodology to validate the expression of schizotypy, including the experience of psychotic–like symptoms, in daily life.[63]

Criticisms of the Construct of Schizotypy

The processes of submitting manuscripts for peer review, presenting at psychology, psychiatry, and personality conferences, and late night conversations with colleagues have provided us with the opportunity to consider many criticisms about the construct of schizotypy and research methods used to assess it. We have attempted to summarize and respond to a sampling of the recurring concerns that we have encountered (some of which we believe have merit, others which may represent important misconceptions to be addressed).

Criticism #1: Schizotypy Does Not Exist

The initial criticism is not focused on the research method or findings, per se, but instead questions the validity (or even the very notion) of the construct of schizotypy. A pair of prominent psychosis-proneness researchers commented that when they presented their initial findings at a psychiatry meeting in the early 1980's, they were told that "people either had schizophrenia or they didn't—and that there was no middle ground." Whereas schizophrenia was originally the province of psychiatry, schizotypy arose from psychology, and literature searches suggest that schizotypy was much more readily incorporated in the field of psychology than in psychiatry. Tsuang[64] stressed the importance of expanding the concept of schizophrenia to include schizophrenia-spectrum disorders, as well as nonclinical manifestations of schizotaxia. In fact, Tsuang et al[65] adapted Meehl's term schizotaxia to describe the expression of neurodevelopmental vulnerability for schizophrenia—analogous to the use of schizotypy. Currently, most neurodevelopmental models of schizophrenia either explicitly or implicitly recognize that the vulnerability for schizophrenia is expressed across a continuum[66]—consistent with the formulation of schizotypy.

Criticism #2: There Is Not a Clear A Priori Model of the Multidimensional Structure of Schizotypy

As noted above, the heterogeneity of schizotypy appears to be represented in a multidimensional structure; however, the historical models of schizotypy did not articulate such a factor structure and tended to refer to schizotypy as an omnibus construct. Furthermore, whereas most current measures assess schizotypy in a multidimensional fashion, in many cases this factor structure was the result of exploratory, post hoc examinations, not a priori conceptualizations. Thus we have a situation in which researchers often acknowledge and assess schizotypy as multidimensional, but their conceptual understanding of this structure may be driven more by the choice of their measure than by an a priori conceptual model driving the development and choice of measures.[67] We suggest that the utility of schizotypy depends upon the development of a clear theoretical model that articulates its multidimensional structure.

Criticism #3: The Study of Schizotypy Is Largely Limited to Research Using Psychometric Inventories and Involving College Student Samples

Psychometric assessments have been widely (and effectively) used to assess schizotypy, often in conjunction with other questionnaire, interview, and laboratory measures. Nevertheless, a number of legitimate criticisms can be leveled at these measures: as noted above many of these scales (and their multidimensional structures) do not adequately tap current conceptualizations of schizotypy, the measures have different multidimensional structures and purportedly comparable factors across measures often are measuring different constructs, almost none of these measures were developed with modern measurement models such as item response theory and their psychometric properties are not always ideal.[67] However, psychometric inventories are relatively inexpensive and noninvasive, and they provide a useful method for "getting our foot in the schizotypy door" and a promising tool for aiding in the identification of biological markers. Thus the psychometric method seems sound, but there is a need to develop new measures based upon current conceptual models of schizotypy and employing modern measurement tools. Regarding the second part of the criticism, the study of schizotypy (and especially the psychometric study) has often relied on college student samples. A recent and notable exception is the longitudinal research by Blanchard and colleagues[45] examining social anhedonia in a community sample. Critics suggest that the use of so-called "analog samples" is invalid because college students have a lower risk for developing schizophrenia. However, as Lenzenweger[24] points out, schizotypy is not simply an analog of schizophrenia, but presumes to indicate carriers of genuine liability. Note that the current model of schizotypy (and data from longitudinal studies) implies that there is meaningful variance associated with schizotypy that can be measured in college student samples. If we hypothesize a continuum of schizotypy anchored by schizophrenia at the extreme end, we would expect that comparable (albeit milder and transient) expressions of schizophrenic phenomenology will be expressed across the continuum—and that we can predict and measure this variation across the continuum (including in college student samples). In a sense, the use of these samples presents a conservative approach, given that they are expected to have an excess of protective factors and to include future patients with relatively good premorbid adjustment (as this ascertainment strategy generally fails to capture schizotypes with early onset and poor premorbid functioning). The successful findings with college student samples encourage extending this method to screening broader samples and integrating it with clinical and family risk studies.

Criticism #4: Longitudinal Studies of Schizotypy Fail to Identify Individuals Who Transition into Schizophrenia at an Appreciable Rate

This is a common misconception that we suggest is twice erroneous. First of all, it appears to be based upon an invalid assumption that schizotypy is equated with the prodrome and incipient transition into psychosis. On the contrary, the present operationalization of the construct implies that most schizotypes are not expected to decompensate into spectrum disorders. As noted above, Meehl speculated that only about 10% of schizotypes will transition into schizophrenia. We do not offer Meehl's speculation as an epidemiological standard, but rather as an illustration that although schizotypy conveys risk for schizophrenia and related disorders, it (thankfully) does not invariably result in psychiatric illness. It also means that schizotypic individuals who remain

compensated are of just as much scientific import as their decompensated peers in that they may inform us about protective factors.

Criticisms about transition rates also fail to fully examine the extant data. An unnamed colleague once mentioned that it was a shame that the Wisconsin[47] longitudinal study "only found a 5% rate of psychosis at the follow-up." We suggest that there are two problems with this conclusion. First of all, this rate was found in a high-functioning sample who at an age of 30 were only mid-way through the window of risk for developing schizophrenia. Secondly, when multiple predictors were considered, the rate of psychosis increased dramatically (eg, in the Chapman et al study, 40% of participants identified by the combination of the Magical Ideation and Revised Social Anhedonia Scales, and who exhibited moderate psychotic–like experiences at the initial assessment met criteria for psychotic disorders at the follow-up assessment).[47] Likewise, Kwapil[48] reported that a quarter of his 30-year-old sample identified solely by social anhedonia met criteria for schizophrenia-spectrum disorders. Strikingly, these rates are higher than those typically found in first degree relatives of schizophrenic patients.

A related criticism is that schizotypy studies, and particularly those employing the psychometric method, are beset by a serious problem with false positives. Obviously, this issue depends in large part on the construct being measured. Again, if the target is schizophrenia or incipient illness, the method does result in many false positives. However, if the target is schizotypy in line with the conceptualizations drawing on Meehl or Claridge, this remains an open question—open in large part because we lack a gold standard and must rely on construct validation of an open construct. In fact, this criticism seems to confuse construct validity (appropriate in the case of schizotypy), with a diagnostic–based criterion validity (seemly more suitable in the case of prodromal cases who are on the brink of schizophrenia). Unfortunately, construct validity demands more patience to formulate and test hypotheses about the construct than does criterion validity. For example, in the Wisconsin follow-up study, between 5% and 40% of the at-risk samples were psychotic at age 30. What does this mean about the remaining 60–95% of the sample? Lacking a crystal ball (and further longitudinal assessments), we do not know if they are false positives, if they are schizotypes who will decompensate in the future, or if they are schizotypes who are and will remain compensated (likely due to protective factors that we fail to recognize at this point). Thus we strongly advocate for continued longitudinal studies of existing samples, as well as the development of new longitudinal studies to examine both risk and resilience in schizotypic young adults. Furthermore, it would be profitable to examine schizotypy and risk for schizophrenia-spectrum disorders in the offspring of schizotypic participants (such as the children of the participants in the Chapmans' longitudinal study).

Where Are We Now, Where Should We Go?

The descriptive psychopathology tradition, traditional diagnostic nomenclature, prodromal models, psychosis-proneness models, and the personality psychology literature, all support the idea of a broader continuum of schizophrenia-spectrum psychopathology. Schizotypy provides a promising, useful, and integrative construct for capturing pathological and subclinical variation across this continuum. Furthermore, we believe it offers an important tool for increasing the power of genetic studies and for enhancing the search for candidate endophenotypes. We suggest that the blueprint for advancement of this construct should include: clearer articulation of the

theoretical model of schizotypy (which we believe is an important goal of this special section), better definition of the multidimensional structure of schizotypy, need for the development of better measures that reflect current conceptual models and employ modern measurement techniques, and the need to resolve dimensional vs taxonic debate. We believe that the starting point for moving forward is to develop a clear and comprehensive operationalization of schizotypy. The number of published studies on schizotypy is dramatically increasing, but these studies all too often either lack a clear operationalization of the construct or have conflicting characterizations. We have offered a brief description in this overview, but a more comprehensive model that characterizes etiology, development, phenomenology, and associations with related constructs is essential. As noted previously, we believe a strength of the schizotypy construct is that it unifies a broad range of conditions. Obviously a consensus model would be powerful, but we believe that clearly operationalized competing models would also energize the field. The bottom line is that such theoretical models should provide the basis of measurement and construct validation. Given the development of clear and testable models, the next step is to develop measures that are tightly linked to these models. We are strong advocates that psychometric assessment provides a promising point of entry for assessing schizotypy and strongly recommend that new questionnaire and interview measures should follow from these new conceptualizations. Given these conceptual and measurement advances, we would argue for an integrative research strategy that examines developmental processes across the range of subclinical and clinical expression of schizotypy. Specifically this would focus on: (1) identification of candidate genes and their mode of action in schizotypy and risk and resilience for clinical disorders, (2) identification of environmental factors (eg, stress, trauma, attachment) and gene × environment interactions underlying schizotypy and the development of clinical disorders, (3) identification of cognitive, neurological, and behavioral endophenotypes that characterize schizotypy broadly defined, (4) identification and differentiation of the processes underlying the positive, negative, and disorganized dimensions of schizotypy with a goal of developing interventions that minimize the likelihood of the development of clinical disorders and ameliorate impairment caused by clinical conditions. Again, we believe that schizotypy provides a unique, useful, and unifying construct for understanding the origins and development of schizophrenic psychopathology.

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